Intensive care management of patients with severe intracerebral haemorrhage after endovascular treatment of brain arteriovenous malformations

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Abstract: We studied the impact of emergency neurosurgery and intensive care on the outcome for patients with severe intracerebral haemorrhage after endovascular treatment of brain arteriovenous malformations (AVMs). We reviewed the case notes of 18 patients with severe haemorrhage after embolisation of a brain AVM between 1986 and 2001. During this period the treatment changed: before 1993, these patients were not surgically treated, and they died, while after 1994, all patients underwent emergency surgery. We established a standardised protocol for emergency treatment and intensive care in May 1998, and emergency surgery was performed as soon as possible after the onset of symptoms of haemorrhage. Postoperative intensive care was according to a standardised regime. During these 15 years, 24 out of 605 patients undergoing 1066 interventions had a haemorrhage during or after the procedure, of which 18 were severe (3% of patients, 1.7% of interventions). All patients had a severe clinical deficit (mean Glasgow coma scale 4.2); eight had uni- or bilateral mydriasis. From 1989 to April 1998 four (31%) of 13 patients died, one (7.5%) remained in a vegetative state and eight (61.5%) made a good recovery. All five patients treated between 1998 and 2001 had a favourable outcome. The mean time from onset of the symptoms of haemorrhage to reaching the operation room was 129 min between 1989 and 1998 and 24 min between 1998 and 2001. Standardised emergency treatment and intensive care with early resuscitation, minimal radiological exploration before rapid surgery improved the outcome. A short time between the onset of the symptoms of haemorrhage and evacuation of the haematoma may be the most important factor for a favourable outcome.

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Intensive care management of patients with severe intracerebral hemorrhage after endovascular treatment of brain arteriovenous malformations

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Abstract

The impact of emergency neurosurgical and intensive care treatment on the outcome of patients with severe intracerebral hemorrhage after endovascular treatment of brain arteriovenous malformations (AVMs) is evaluated. The Charts of 18 patients suffering from severe intracerebral hemorrhage after embolization of brain AVMs between 1986 and 2001 are retrospectively reviewed. During these consecutive periods the treatment changed. Before 1993 patients with severe intracerebral hemorrhage were not surgically treated and died. After 1994 all patients underwent emergency surgery. In May 1998 a standardized protocol for emergency and intensive care treatment was established and surgical emergency treatment was performed as fast as possible after symptom onset of hemorrhage. Postoperative intensive care treatment was performed according to a standardized regimen. During this 15 years in 605 patients with 1066 interventions 24 per- or postprocedural hemorrhages occurred. Among them were 18 patients who suffered from severe hemorrhages (2.97% of patients; 1.69% of interventions). All patients showed a severe clinical presentation (mean Glasgow Coma Scale 4.2), eight patients had unilateral or bilateral mydriasis. During the time period from 1989 to April 1998 four of 13 patients died (31%), one patient remained in a vegetative state (7.5%) and eight patients recovered with good outcome (61.5%). Between 1998 to 2001 all five patients survived and recovered with favorable outcome. The time interval from onset of the symptoms of hemorrhage to reach the operation room for the surgical intervention was between 1989 and 1998 129 mins., and between 1998 and 2001 24 mins. Standardized emergency and intensive care treatment with early resuscitation, minimal radiologic exploration before rapid surgical treatment improve the outcome in patients with severe intracerebral hemorrhage after endovascular treatment of brain AVMs. A short time interval between the onset of the symptoms of hemorrhage and the surgical hematoma evacuation may be the most important factor in predicting a favorable outcome.

Key words:

Intracerebral hemorrhage; Embolization; Arteriovenous malformation; Intensive care treatment

Introduction

The most serious complications related to the endovascular treatment of arteriovenous malformations (AVMs) are cerebral ischemia and hemorrhage. Severe perprocedural and early postprocedurale hemorrhages are described to occur in 3.1% to 11% of the patients after AVM embolization [2, 3]. So far literature dealing with aspects of intensive care treatment in patients after an AVM embolization induced hemorrhage is sparse. We report on our experience with 18 cases of life threatening intraparenchymatous and intraventricular hemorrhage that occurred during or within a few days after AVM embolization. The role of emergency neurorescucitation is focused and guidelines for neurointensive care treatment are given.

Methods

Between 1986 and 2001 605 patients with 608 AVMs underwent 1066 endovascular treatment sessions. The charts and imaging studies of 18 patients with perprocedural and early postprocedurale severe hemorrhage within seven days after AVM embolization were reviewed. Severe hemorrhage was described as the patient developing severe neurological deterioration with space-occupying intraparenchymatous and/ or intraventricular bleeding. The goal of embolization was the obliteration of the AVM-nidus following superselective microcatheterization of the identified feeding arteries and angiographic analysis of its intrinsic vascular architecture [1]. The main embolic material used was cyanoacrylate. Until 1991 embolization was performed with IBCA and afterwards with NBCA (Histoacryl, Braun, Melsungen, Germany). Retardation of polymerization of the NBCA was achieved with the addition of variable amounts of Pantopaque (Lipiodol), depending on the type of intranidal av-shunts. In 30% of cases micropaticles of polyvinylalcohol (PVA) were used as a supplementary embolic material to complete obliteration of the AVM following obliteration of the main portion of the nidus with NBCA. In all patients the guiding catheters used during the procedure were continuously flushed with heparinized saline (2000 U/L) but no additional systemic heparinization was used throughout this series.

During the consecutive time periods between 1986 and 2001 several treatment aspects changed: During the first period most patients were treated under local anesthesia. Because of several advantages, since 1993 all patients underwent embolization under general anesthesia [2]. Following the procedure and removal of the femoral catheter or sheath, all patients were extubated, examined neurologically and transferred to the intermediate care or neurocritical care unit for close bedside monitoring for 12 to 24 hours before transfer to the ward. Before 1993 three patients with signs of massive bleeding and imminent brain herniation were not surgically treated with hematoma evacuation and died. After 1994 all patients with a severe intracerebral hemorrhage were treated surgically with hematoma evacuation and/or ventricular drainage. The treatment of elevated intracranial pressure (ICP) in the earlier years was limited to ventricular drainage and the application of mannitol 20% and thiopenthal boluses. In May 1998 a standardized protocol for emergency and intensive care treatment was established. Patients with clinical suspicion for hemorrhage after neuroradiological interventions were continuously supervised and treated by specialists for neurointensive care until they were transferred to the operating room. Following the treatment protocol, rapid clinical deterioration and high clinical suspicion for hemorrhage led to an immediate emergency resuscitation with induction of anesthesia (etomidate 0.3 mg/kg intravenously, combined with rocuroniumbromid 0.8 mg/kg and with clinical signs of imminent brain herniation combined with thiopental 3 – 5 mg/kg), endotracheal intubation and cardiovascular stabilization. Acute elevated ICP was treated with 100ml of mannitol 20% and/or 100ml hypertonic NaCl-hydroxyethyl-starch solution (Hyper-HES) (2570 mosm/l). Thiopental boluses were given in a loading dose of 10mg/kg and continued in a dosage of 5mg/kg/hour. An emergency CT scan was carried out immediately. If a cerebral hematoma with mass effect and/or intraventricular bleeding was diagnosed the patient was transferred immediately to the operating room for emergency craniotomy. The hematoma was evacuated, which provided brain decompression and relaxation. Any residual AVM was excised, using standard microvascular techniques. At the end of the procedure, an ICP monitor (ventricular drainage or subdural probe) was placed in all patients. Postoperative sedatives (fentanyl infusion 5 - 8 ug/kg/hr and midazolam 0.1 – 0.4 mg/kg/hr) were not reduced before ICP remained below 15 mmHg for at least 24 hours and a postoperative CT scan control was performed. After a loading dose of 10mg/kgBW the dosage of barbiturates was postoperatively adapted to a burst suppression pattern in continuous EEG-monitoring. If ICP elevations > 15mmHg occurred, the CT scan control was performed earlier to exclude rebleeding. The target values for blood pressure management were readapted to reach cerebral perfusion pressure (CPP) values above

60 mmHg. Elevated ICP was treated with ventricular drainage and/or by continuing barbiturate coma to obtain burst suppression on EEG and/or mild hypothermia (body core temperature 33 to 34°C) [4]. In all patients with elevated ICP, two combined fiberoptic thermistor catheters were placed in the internal jugular vein and in the femoral artery in order to monitor the jugular bulb saturation and to measure the cerebral blood flow (CBF) [5] [6]. Final outcome was assessed after one year and late outcome in two patients after 3 and 6 months according to the Glasgow Outcome Scale (GOS) [7].
The mean size of hemorrhage in different patient groups were compared with unpaired t-test.

Results

In the course of these 15 years in 605 patients with 1066 interventions 24 per- or postprocedural hemorrhages occurred (3.97% of patients; 2.25% of interventions). In six patients the hematomas were small (less than 2.5cm in maximal diameter) and mostly asymptomatic or accompanied by headaches but no neurological deficits and were discovered incidentally on routine postembolization MRI. In 18 cases the patients suffered from severe hemorrhages with neurological deterioration (2.97% of patients; 1.69% of interventions). 13 hemorrhages occurred between 1986 and April 1998 and five between May 1998 and April 2001. Patient characteristics are given in table 1.

Table 1

There were 11 male and seven female patients with a mean age of 41 years. All patients showed severe clinical presentation (mean Glasgow Coma Scale 4.2 before intubation), eight patients had unilateral or bilateral mydriasis. Three types of clinical presentation could be differentiated. Nine patients (group one) showed an acute loss of consciousness whereas five patients (group two) slowly deteriorated over one or two hours. A third group with three patients showed increasing drowsiness, in two patients associated with clinical signs of hydrocephalus (headache, vomiting). The CT scans from these latter patients showed isolated intraventricular bleeding without intraparenchymatous hemorrhage. Two patients were treated with ventricular drainage and one patient recovered spontaneously over several days. In group one and group two, CT scans showed large intracerebral hemorrhages. The mean size of

hemorrhage in group one tended to be higher (mean 5.8 cm) than the maximal diameter of hemorrhage in group two (mean 5 cm) without statistical significance (p = 0.087).
15 patients were treated surgically with hematoma evacuation, in three patients combined with ventricular drainage. Three patients with ventricular tamponade were treated with ventricular drainage only. In six patients the AVM could be totally excised during the surgical emergency treatment.
The main results for the patients of the two periods before and after standardized emergency and intensive care treatment are given in table 2.

Table 2

During the time between 1989 and April 1998, 10 out of 13 patients were treated surgically, between May 1998 and 2001 all five patients were treated with hematoma evacuation and/or ventricular drainage. On the whole four patients died (22%), one patient remained in a vegetative state (6%) and 13 patients (72%) survived with a favorable outcome (GOS 4 and 5). During the period between 1989 and April 1998 4 out of 13 patients died (31%), one patient remained in a vegetative state (7.5%) and eight patients recovered with good outcome (61.5%). Since May 1998 the outcome improved with introduction of standardized emergency and neurointensive care treatment. All five patients survived and recovered with favorable outcome. The mean time interval between the onset of symptoms of hemorrhage and the surgical intervention in patients treated in the early years was 129 mins. (range 30 to 480 mins.) and was significantly longer compared to the time interval of 24 mins. (range 20 to 30 mins.) for patients who were treated after May 1998.
In the five patients treated since May 1998 the mean duration of the ICU stay was 8.2 days (range 5 to 11 days). In two of the five patients ICP increased to values above 15 mmHg in the postoperative period. In both patients a retrograde fiberoptic jugular vein catheter for monitoring cerebral hemodynamics was added. Elevated ICP in both patients was treated with ventricular drainage, in addition in one patient the barbiturate coma was continued for two days.

Discussion

General considerations

Hemorrhagic events represent the most life-threatening complication that is associated with embolization of AVMs. Embolization associated hemorrhage may occur either during the procedure (perprocedural hemorrhage) or during the early postembolization period. Perprocedural hemorrhage may be caused by mechanical perforation of a cerebral artery during microcatheterization or due to hemodynamic changes induced by partial embolization of the nidus [2, 3]. Early postembolization hemorrhage is caused by hemodynamic changes occurring in the hours following partial or subtotal embolization of the AVM, mostly by impairment of the venous drainage of the AVM [2, 8]. The three types of clinical presentation in our patients may lead to the conclusion that perprocedural hemorrhage may originate from three different mechanisms. In the nine patients with hyperacute loss of consciousness tending to larger intraparenchymatous hemorrhages in CT imaging (group one) venous congestion may cause a rupture in one of the arterial feeders or in the nidus itself at the arterio-venous junctions. On the other hand the slow deterioration in the five patients in group two with smaller size of hemorrhages may assign a venous type of bleeding. The third group of three patients showing increasing drowsiness with clinical signs of hydrocephalus is associated with isolated intraventricular bleeding.

Since May 1998 the treatment of patients with complications after neuroradiological interventions was taken over by the specialists for neurointensive care until they were transferred to the operating room. A standardized protocol for emergency and intensive care treatment was applied. According to this protocol patients underwent emergency resuscitation with immediate intubation, minimal radiologic exploration with a diagnostic CT scan and acute treatment of elevated ICP. Time consuming procedures not immediately required for the treatment were strictly avoided in order to minimize the time interval between the onset of the symptoms of hemorrhage and the surgical hematoma evacuation. With this strategy since May 1998 all five patients reached the operating room within 30 mins., hence in significantly shorter time than in the earlier years. The good functional outcome of all five patients in this recent period compares favorably with the outcome in other, previously reported series. The difference may be explained mainly by the very short delay between abrupt neurological

deterioration and surgical decompression in our patient population since May 1998. In the series of Valavanis and Yasargil from 1986 to 1996 with 387 patients and a total of 710 embolization sessions, 9 cases with severe per- and postprocedural hemorrages occurred (2.3%). Three of these patients died because of massive hemorrhage and brain herniation. Six patients underwent surgical emergency removal of the hematoma because of rapidly progressing clinical deterioration. Of these, two had a good outcome (22%), three a moderate and one a poor outcome (45%) [2]. In the series of Purdy et al. seven of 63 patients with cerebral AVMs treated with embolization developed intracranial hemorrhagic complications. Two of these seven patients died (29%), one had a significant residual neurological impairment (14%), and the other four developed no significant residual impairment (57%). The authors concluded that immediate surgical intervention can yield gratifying outcomes in potentially catastrophic situations [3]. Jafar reported 10 cases requiring surgical emergency treatment because of a profound neurological deterioration from an AVM hemorrhage [9]. Eight patients had a hemorrhage secondary to an embolization procedure. Most patients underwent surgery within 30 minutes of the ictus. Nine patients had an good-to-excellent outcome (90%) and one patient remained hemiplegic (10%). The authors concluded that prompt hematoma evacuation with simultaneous AVM excision as well as perioperative ICP control with mannitol and barbiturates can yield a good to excellent outcome [9].

Implications for emergency resuscitation and postoperative intensive care

The standardized procedure given in table 3 allowed rapid evaluation and treatment:

Table 3

Emergency evaluation: Typical symptoms of AVM bleeding are: Headache, dizziness, vomiting and hemiparesis with slow deterioration (venous or intraventricular bleeding type) or hemiplegia with hyperacute loss of consciousness and mydriasis (arterial type). With high clinical suspicion for hemorrhage, emergency CT scan should be immediately organized and the neurosurgeon should be informed immediately as well.

Basic intensive care

includes endotracheal intubation, the insertion of central venous and arterial catheters and blood samples for electrolytes and coagulation. If patients are treated with heparin, 1000 U

protamine is given as an emergency reversal dose. Blood samples for ACT (activated clotting time) and PTT (partial thromboplastin time) are taken to fine-tune the final protamine dose. Rapidly decreasing consciousness with aspiration and respiratory depression require immediate intubation and mechanical ventilation. Cardiovascular instability has to be avoided carefully. Rises of blood pressure provoke rebleeding and hypotension may compromise cerebral perfusion. Therefore sedatives for intubation should be sufficiently dosed and have minimal cardiovascular side effects (for example etomidate 0.3 mg/kg). With clinical signs of imminent brain herniation thiopental 3 – 5 mg/kg with its ICP-lowering properties may be preferred as sedative for intubation. Hypotension after thiopenthal can be avoided by previously correcting hypovolemia. Hemodynamic response to intubation may be attenuated if sedatives are supplemented by a short-acting neuromuscular blocking agent (for example rocuronium 0.5 mg/kg) [18]. Coughing and cardiovascular instability during transport maneuvers have to be carefully avoided. Therefore deep sedation and neuromuscular relaxation should be maintained until the surgical hematoma evacuation can be performed.

Treatment of acute elevated ICP:

Medical treatment of increased ICP in the emergency setting includes mild hyperventilation with controlled mechanical ventilation [11]. The arterial PCO_2 is kept between 28 and 33 mmHg. Endexpiratory CO_2 concentrations have to be monitored during the transport procedures. Hyperventilation to low $PaCO_2$-values may reduce CBF below ischemic thresholds and produce secondary brain damage [11]. Therefore hyperventilation without CBF-monitoring should only be used as a powerful tool for achieving a short-term reduction in ICP until decompressive surgery and hematoma evacuation can be performed. Mannitol 20% 100ml and/or hypertonic NaCl-hydroxyethyl-starch solution (Hyper-HES) are effective in acute treatment of elevated ICP [12] and may improve diminished CBF [13]. During osmotherapy plasma osmolarity (target range for plasma osmolarity 300-310 mosm/l), serum electrolytes, hydration and coagulation parameters are to be carefully controlled [14]. Thiopental boli are given in a loading dose of 10mg/kg. Barbiturates reduce the cerebral blood volume with subsequent ICP-reduction [15]. Nevertheless, the side effects, such as hemodynamic instability in the acute phase and the increased rate of pulmonary infections in postoperative intensive care, have to be kept in mind [16]. The goal of a minimal CPP > 60mmHg can be achieved by adequate volume expansion and vasoactive drugs (for example norepinephrine or epinephrine 5-10 µg/min). During anesthesia in the operation room secondary brain damage may be prevented by giving a continuous dosage of thiopenthal (5mg/kg/hour) as free-radical savengers and by reducing cerebral metabolic requirements of

the brain [17]. Furthermore barbiturates may be helpful to sustain controlled hypotension during hematoma evacuation and resection of the residual AVM.

Cardiovascular stabilization:

Based on their clinical experience Young and Pile-Spellman recommend while acute bleeding is occurring, the blood pressure to be kept as low as possible [18]. Using a biomathematical AVM model, based on electrical network analysis Massoud et al. found that the induction of systemic hypotension can prevent nidus rupture during and after AVM embolization [19]. Hypertension should be strictly avoided because of possible bleeding from a partially open nidus. On the other hand, to maintain CBF adequate CPP should be maintained. Nevertheless, during and immediately after AVM embolization induced hemorrhage judicious blood pressure control and a careful balance between maintaining cerebral perfusion and minimizing the risk of rebleeding is mandated.

CT scan:

CT is the imaging modality of choice to detect acute intracerebral hemorrhage from ruptured AVM. Most commonly, this occurs in the brain parenchyma adjacent to the AVM. Subarachnoid or intraventricular hemorrhages are less frequent [2]. In patients with AVMs, subarachnoid hemorrhage is most probably caused by the rupture of an associated proximal aneurysm and not by AVM rupture.

Postoperative Intensive Care:

All patients are maintained under general anesthesia for the following 24 hours after surgery. It is obvious, that systemic factors aggravating brain edema and secondary brain damage have to be treated immediately and consequently [20]: Hypoxia, hypercapnia and hypocapnia must be strictly avoided. The electrolyte balance has to be maintained. Hyponatriemia worsens brain edema and must be corrected. Infusion of hypotonic solutions in patients at risk for brain edema has to be avoided. Hyperglycemia at the time of stroke is associated with poor outcome [21]. Therefore blood glucose concentration should be strictly kept within a range of 5.5 - 8 mmol/l. Hyperthermia increases the cerebral metabolic rate and worsens the outcome after an acute stroke [22] [23]. Employing antipyretics and cooling devices should rigorously avoid fever. The patient should be maintained in a 15° to 30° upright position without causing jugular obstruction by bending the neck or rotating the head. Recently, head elevation has become controversial because of reduction in carotid artery pressure, with subsequent decreased CPP. The ideal posture is found individually if ICP and CPP monitoring is available. Increases of blood pressure are to be avoided after microsurgical AVM resection. In some instances, especially if the residual AVM is partially resected, controlled mild to

moderate hypotension is desired to prevent rebleeding. In this specific setting, to guarantee adequate cerebral perfusion, ICP monitoring in the postoperative setting is essential. Critical drops in the perfusion pressure may have undesirable ischemic consequences in the precarious surrounding of the parenchyma in which autoregulatory mechanisms have already resulted in maximum arterial dilatation [24]. This could also predispose to slugging, aggregation, and clumping platelet aggregates and red blood cells in the venous outflow system [8]. To sustain mild controlled hypotension and to prevent ICP-elevations patients remain sedated for the following 24 hours after surgery. The dosage of babiturates, started during emergency resuscitation and continued during the surgical intervention, is postoperatively adapted to a burst suppression pattern in continuous EEG-monitoring. Cardiac output, pulmonary capillary wedge pressure or recently available volumetric systemic hemodynamic parameters like intrathoracic blood volume are monitored using pulmonary artery or special femoral artery thermodilution catheters [25]. Heart failure has to be expected with large midline AVMs that drain into an enlarged vein of Galen. Sedatives are not reduced before ICP remains below 15 mmHg for 24 hours and postoperative CT scan is performed. If ICP elevations occur, postoperative CT scan is advanced to exclude rebleeding. The target values for blood pressure management are readapted to reach CPP values above 60 mmHg. Elevated ICP due to brain edema should be treated under extended monitoring of cerebral hemodynamics (monitoring of jugular bulb O_2-saturation, cerebral blood flow or intraparenchymatous O_2 partial pressure) [5] [26]. Elevated ICP due to brain edema may be treated with ventricular drainage, by continuing barbiturate coma to obtain burst suppression on EEG [27] [15] and/or mild hypothermia (body core temperature 33 to 34°C) [27].

Conclusions

Based on our observations with different treatment strategies between 1989 and 2001 emergency neurosurgical and standardized neurointensive care treatment seems to improve the outcome in patients with severe intracerebral hemorrhage after endovascular treatment of brain AVMs. In the recent series after May 1998 the favorable outcome after AVM embolization associated hemorrhage may be mostly due to the short time interval between clinical deterioration and immediate surgical hematoma evacuation, including early neurorescucitation and minimal radiologic exploration. In the postoperative period, continuous measurement of ICP and CPP is of critical importance since the main risk is the development of cerebral swelling after brain decompression. Neurointensive care treatment should be standardized in principles but adapted to the dynamic requirements of each patient.

This policy, requiring close and fast multipidisciplinary collaboration between neuroradiologists, neurosurgeons competent for the emergency treatment and specialists for neurointensive care proved life saving and led to a favorable outcome in all five patients with intracerebral hemorrhage after endovascular treatment of brain AVM since 1998.

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